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Rev Soc Bras Med Trop 49(3):286-291, May-June, 2016
doi: 10.1590/0037-8682-0454-2015

Major Article

High prevalence of plasmid-mediated quinolone resistance determinants in *Enterobacter cloacae* isolated from hospitals of the Qazvin, Alborz, and Tehran provinces, Iran

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Abstract

Introduction: Plasmid-mediated quinolone resistance (PMQR) is a growing clinical concern worldwide. The main aims of this study were to detect *qnr*-encoding genes and to evaluate the clonal relatedness of *qnr*-positive *Enterobacter cloacae* isolates. Methods: A total of 116 *E. cloacae* isolates that were not susceptible to quinolone were obtained from seven hospitals in Tehran, five hospitals in Qazvin, and two hospitals in Karaj (Iran). Bacterial identification was performed using standard laboratory methods and API 20E strips. Quinolone resistance was determined using the Kirby-Bauer disk diffusion method according to the Clinical Laboratory Standards Institute (CLSI) guidelines. PCR and sequencing were employed to detect *qnrA*, *qnrB*, and *qnrS* genes, and clonal relatedness was assessed using the enterobacterial repetitive intergenic consensus (ERIC)-PCR method. Results: In total, 45 (38.8%) and 71 (61.2%) of isolates showed high- and low-level quinolone resistance, respectively, and *qnr*-encoding genes were detected in 70 (60.3%) of them. *qnrB1* [45 (38.8%) isolates] was the most commonly detected gene, followed by *qnrS1* [28 (24.1%) isolates] and *qnrB4* [18 (15.5%) isolates] either alone or in combination with other genes. The results of the ERIC-PCR revealed that 53 (75.7%) *qnr*-positive isolates were genetically unrelated. Conclusions: This study describes, for the first time, the high prevalence of the *qnrB1*, *qnrS1*, and *qnrB4* genes among *E. cloacae* isolates in Iran. The detection of *qnr* genes emphasizes the need for establishing tactful policies associated with infection control measures in hospital settings in Iran.

Keywords: *Enterobacter cloacae*. Enterobacterial repetitive intergenic consensus-PCR. Plasmid-mediated quinolone resistance.

INTRODUCTION

Enterobacter cloacae is a clinically significant gram-negative bacterium that can cause several clinical diseases such as urinary tract infections, bacteremia and sepsis, lower respiratory tract infections, pneumonia, and soft tissue infections[1]. Health care for patients with infections caused by this organism has been associated with high mortality and morbidity, especially among patients admitted in intensive care units (ICUs)[2,3].

Nowadays, quinolones are being frequently used to treat serious infections caused by enterobacteria in hospital settings[4,5]. Extensive and inappropriate use of quinolones and other such antimicrobial agents has increased multidrug resistance in *Enterobacter cloacae* (MDREC) isolates, which complicates and limits the process of antimicrobial therapy[6,7].

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Received 18 January 2016
Accepted 28 April 2016

Resistance to quinolone compounds is often due to chromosomal point mutations in deoxyribonucleic acid (DNA) gyrase and/or topoisomerase IV[8]. However, plasmid-mediated quinolone resistance (PMQR) has also been reported in several parts of the world[9,10,11,12,13,14]. Plasmids carrying *qnr* genes widely vary in size and typically carry multiple resistance determinants[15,16]. Qnr proteins are members of a pentapeptide repeat protein family, which is capable of protecting DNA gyrase and DNA topoisomerase IV from quinolone compounds[17]. For example, QnrB4 is a characterized pentapeptide repeat protein that interacts with DNA gyrase[18]. Antibiotic treatment against infections caused by *qnr*-positive isolates is more complicated because of the remarkable ability of these organisms to develop resistance to different antibiotic classes as well as their high potential for transmitting antibiotic resistance between different bacterial species[19,20]. Three major groups of *qnr* determinants, *qnrA*, *qnrB*, and *qnrS*, are increasingly being identified in clinical isolates of various enterobacterial species worldwide[21]. The first PMQR gene was reported in a *Klebsiella pneumoniae* isolate from Birmingham in 1994[22]. Later, these genes were also reported in